CEREBRAL ISCHEMIC STROKE IN A PATIENT WITH SICK SINUS SYNDROME

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ABSTRACT

Sick sinus syndrome represent one of the cardiac arrhythmias which may cause ischemic stroke by hemodynamic or thrombo-embolic mechanism, often these two mechanisms coexist and potentiate each other. We present the case of the 65 years old male admitted to our clinic for an acute ischemic stoke in the right carotid territory. The Doppler ultrasonography of the cervico-cerebral arteries performed the next day after the admission showed occlusion of the right internal carotid artery. The patient was transferred to the cardiology clinic where he underwent a DDDR pace-maker implantation.
The ultrasonographic exam performed one week later showed repermeabilisation of the right internal carotid artery.

Keywords: sick sinus syndrome, ischemic stoke, arrhythmias, internal carotid artery occlusion

BACKGROUND

Cardiac arrhythmias represent an important cause of ischemic stroke. The two mechanisms by which cardiac arrhythmias might cause transient or permanent focal cerebral ischemia are hemodynamic and thrombo-embolic. Embolism and hypoperfusion oftentimes coexist and potentiate each other. The link between these two factors in the pathophysiology of brain infarction is the impaired clearance of emboli due to low flow states. Arrhythmias sufficiently severe to reduce cardiac output may reduce global cerebral perfusion, and the brunt may fall more severely in the areas with acquired arterial lesion producing focal cerebral ischemia. When most arrhythmias such as paroxysmal tachycardia and complete heart block become symptomatic they produce global ischemia (i.e. syncope). Only arrhythmias which encourage atrial stasis, such as atrial fibrillation and sino-atrial disorders cause systemic and cerebral embolism.

Chronic sino-atrial desease is variously defined, but shoud include at least sinus bradiarrhythmia, regular sinus bradycardia and sino-atrial block with or without sinus arrest. Many patients have also tachycardia, though this is sometimes considered a separate disorder (bradycardia-tachycardia syndrome).

Sick sinus syndrome is a high-risk source of cerebral embolism. The risk is probably greatest for those patients whose conditions evolve into a paroxysmal or chronic atrial fibrillation pattern or those patients who have left atrial spontaneous echocardiographic contrast and decreased atrial ejection force. Patients with sick sinus syndrome may experience cerebral ischemia even after pacemaker insertion. VVI pacing is associated with a higher risk of embolic complications than atrial or dual-chamber stimulation.

CASE STUDY

We present the case of a 65 years old caucasian male who was admitted to our clinic for abrupt onset of muscle weakness on his left side. The patient had medical history of arterial hypertension, paroxysmal atrial fibrillation and flutter, and pulmonary oedema.

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At the admission to our clinic the neurologic exam revealed left homonimus hemianopia, left central facial palsy, left hemiparesis, Babinki reflex on his left side, left tactile inattention and dysarthria, the patient was conscious but somnolent.

The electrocardiogram showed sinusal bradycardia with 36 bpm with prolonged PR interval and negative T waves in the inferior teritory.

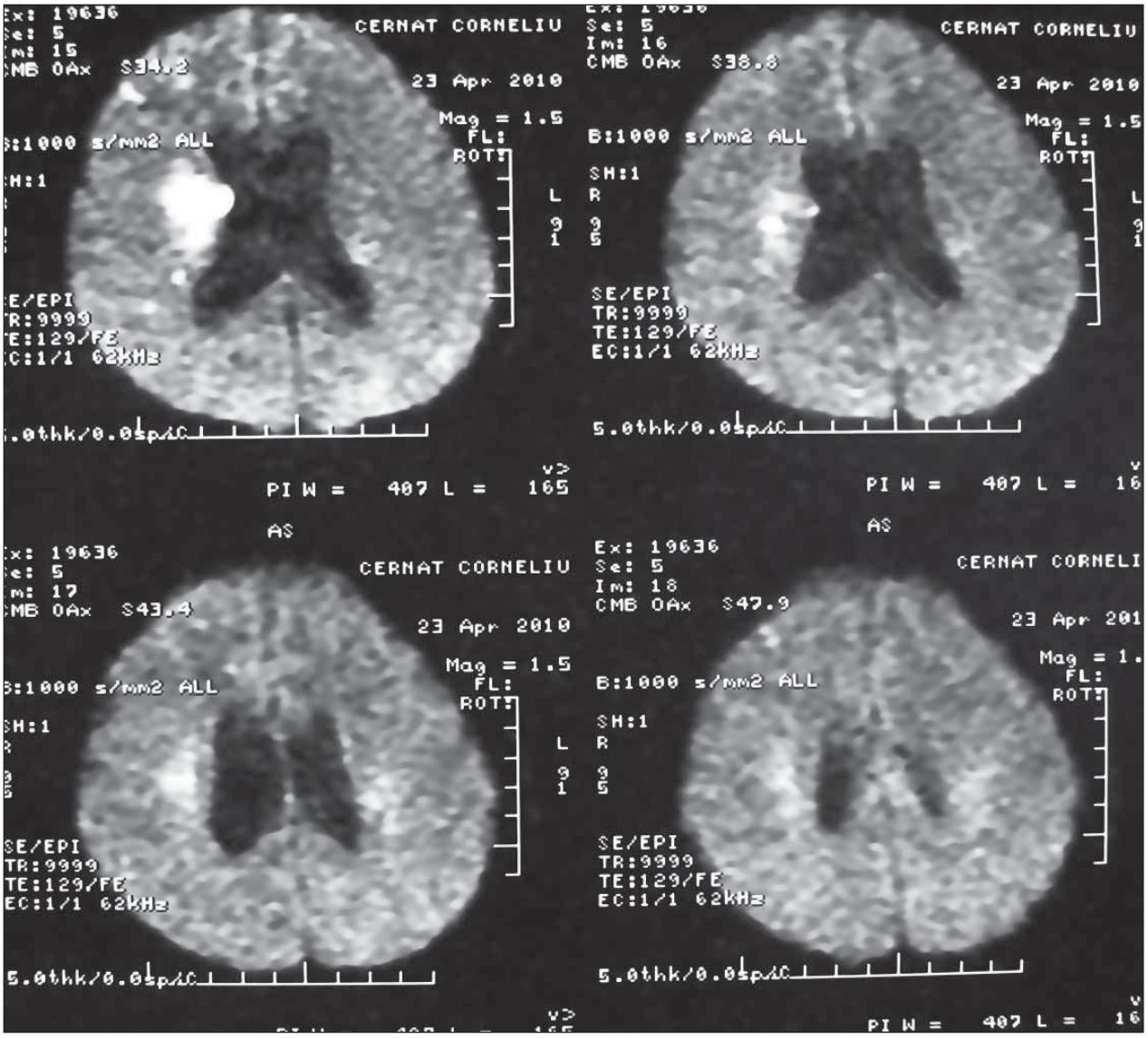
CT scan performed at the admission showed incipient signs of cerebral ischemia in the right middle cerebral artery territory.

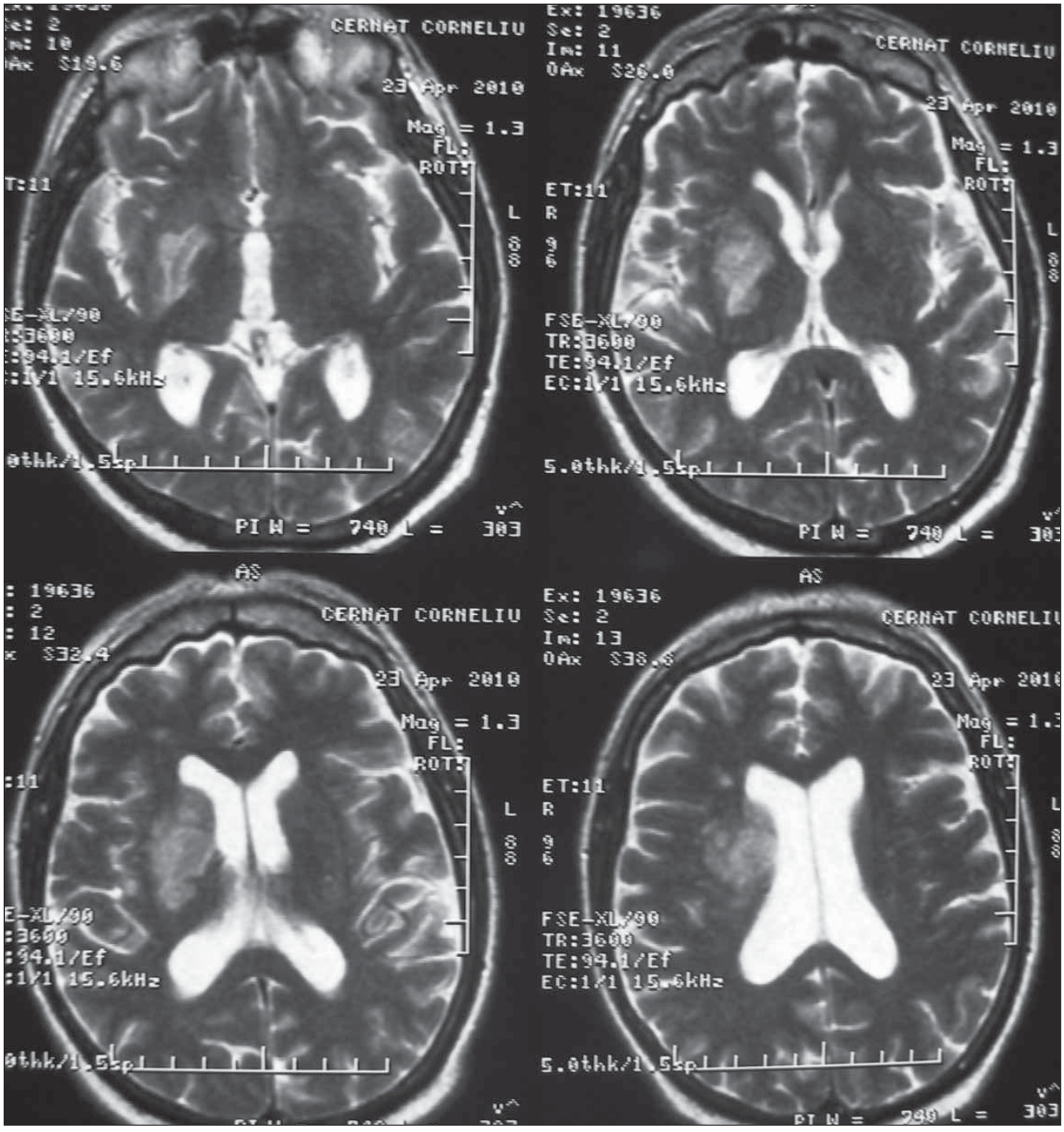
We also performed a cerebral and cervical region MRI with MR-angiography of the cervico-cerebral arteries which showed capsular-lenticular hyperintensity on T2-weighted and FLAIR images with restriction on diffusion-weighted images in the territory of deep branches of right middle cerebral artery with compression of the right lateral ventricle, descreet frontal cortico-subcortical hyperintensity on T2-weighted and FLAIR images with restriction on diffusion weighted images in the border-zone territory. The MRA sequences showed poor flow of the right internal carotid artery in the cavernous part without signs of intraluminal thrombus of the right middle cerebral artery.

The Doppler ultrasonography of the cervico-cerebral arteries performed the next day after the admission showed occlusion of the right internal carotid artery at the origin, produced probably by intra-arterial thrombosis due to hemodynamic changes.

After two weeks of hospitalization the patient was transferred to the cardiology clinic where he underwent a DDDR pace-maker implantation. The transthoracic echocardiography showed mild left ventriclar dysfunction with no evidence of left atrial spontaneous contrast or thrombosis.

The ultrasonographic exam performed one week later showed repermeabilisation of the right internal carotid artery.

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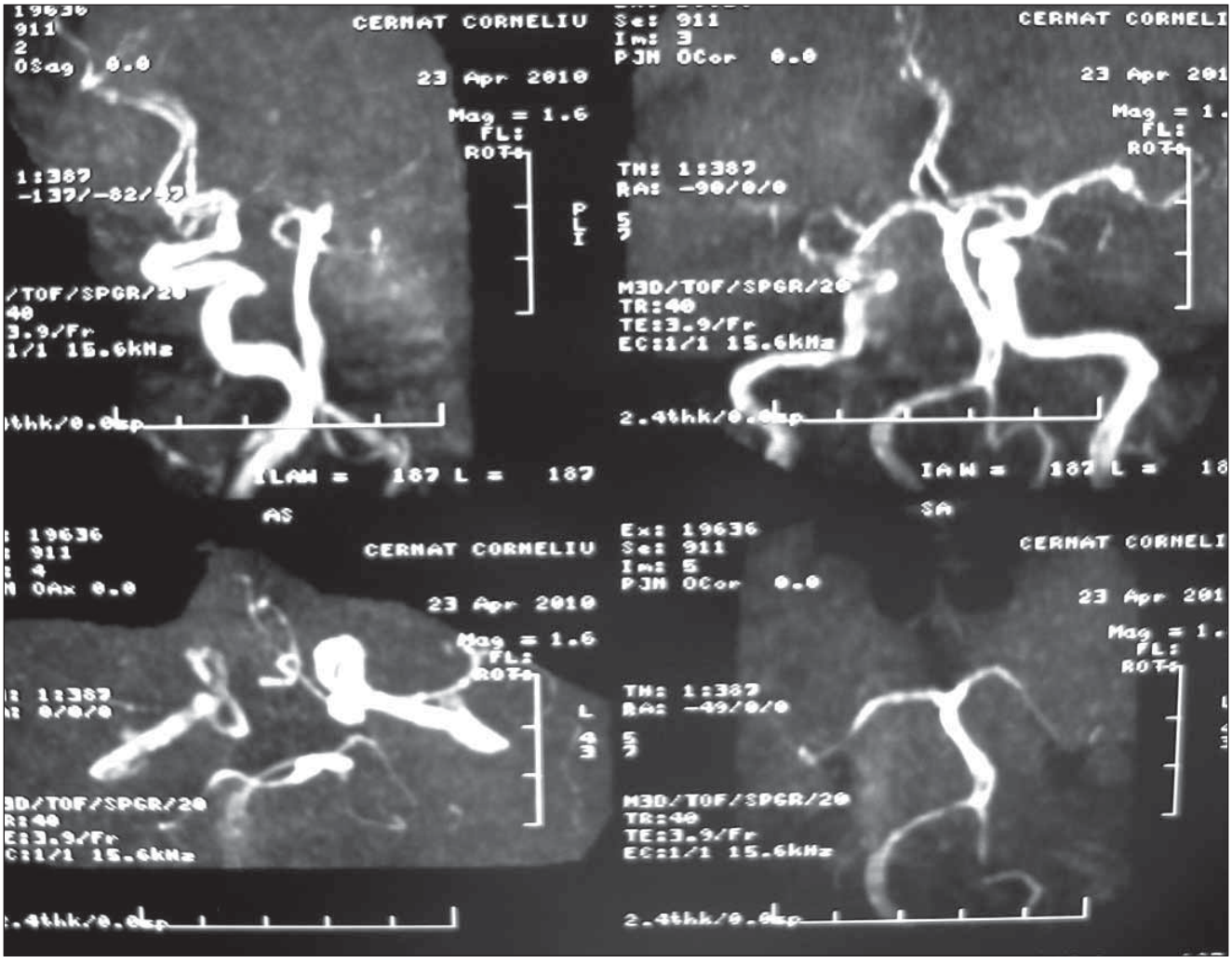
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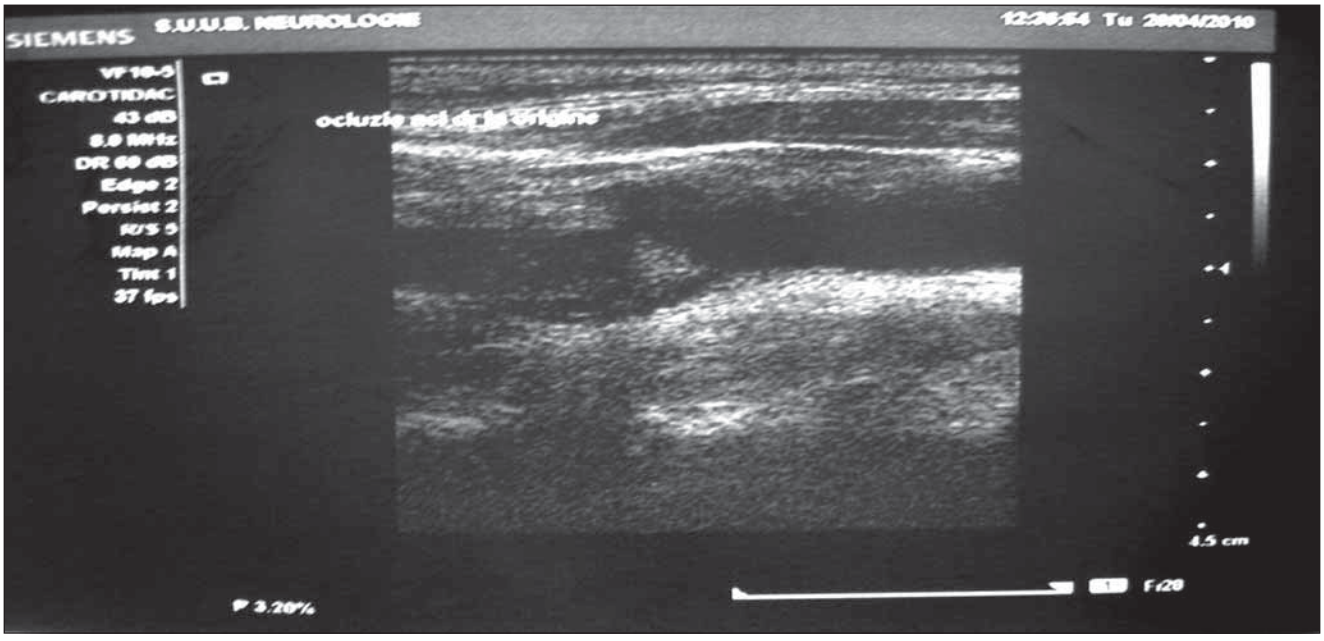

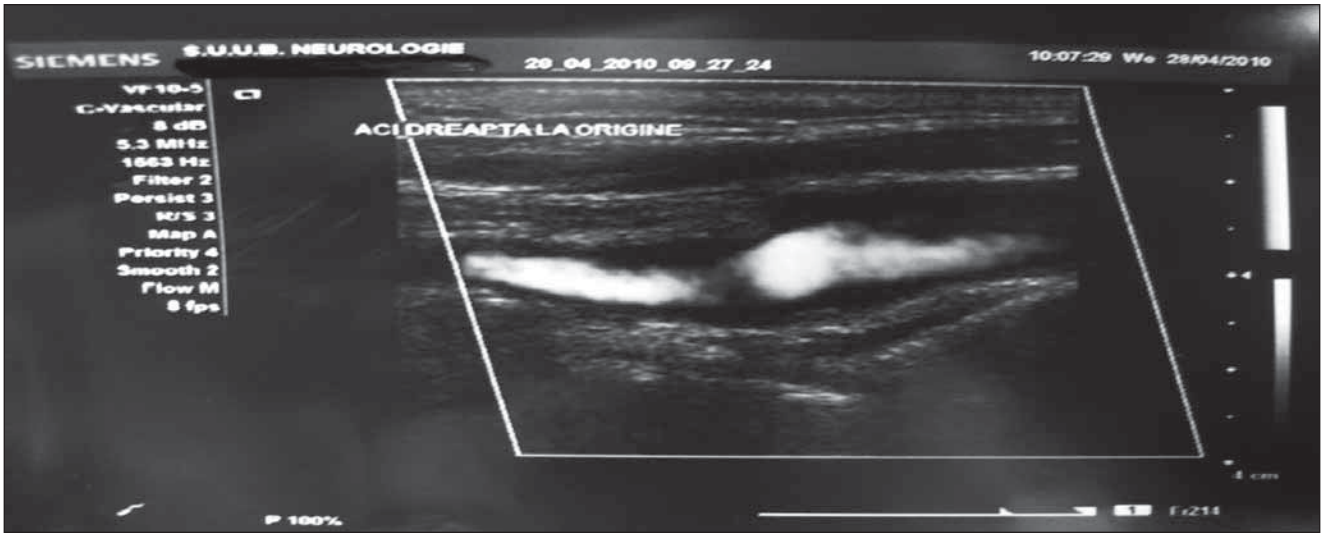

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